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Eighteen months ago members of the Cleveland Clinic Staff formed the Naval Medical Specialists Unit Number 110. Recently the group has been called into active service and has been asked to form a larger unit which will be known as Mobile Naval Hospital Number 4. This will be manned largely by present members of the Clinic Staff or by past members or Fellows. Various Divisions of the Clinic are represented by the present Staff members who formed the original Naval Medical Specialists Unit. Dr. W. James Gardner represents Neurosurgery; Dr. A. C. Ernstene, Cardiorespiratory Diseases; Dr. W. J. Engel, Urology; Dr. E. J. Ryan, Endocrinology and Metabolism; Dr. R. J. Kennedy, Eye; Dr. J. C. Root, X-ray; and Dr. George Crile, Jr., Surgery.

As in other institutions which have sent representatives to the armed forces, the burden of additional work is being felt, especially by the members of the departments named. The present plan is that the practices of those who are now in the service will be carried on by their associates for the duration.

THE DIAGNOSIS AND TREATMENT OF CARDIAC EMERGENCIES

A. CARLTON ERNSTENE, M.D.

Situations in which the prompt institution of proper treatment may be directly responsible for the saving of life probably are encountered more frequently in patients who have organic heart disease than in any other group of individuals presenting medical problems. A thorough understanding of the therapeutic measures available for use in cardiac emergencies, therefore, is of considerable importance. Fortunately, practically all of the measures of established value are of such a nature that one can be prepared at all times to use them.

ACUTE MYOCARDIAL INFARCTION

The most common cardiac emergency is acute coronary artery occlusion with infarction of the myocardium. The clinical picture of this condition is too well known to warrant detailed description. The pain is similar to that of angina pectoris, but is more severe and of longer duration. Symptoms of shock of mild to severe degree usually appear soon after the onset, and fever and leukocytosis generally develop within the first 24 hours. A pericardial friction rub may appear at any time during the first few days after the attack and may last for only a few hours or for several days. The electrocardiogram shows characteristic changes in practically every case if records are made at daily intervals and if both standard and precordial leads are used.

The first aim in the treatment of acute myocardial infarction is to relieve the pain. For this purpose aminophyllin is administered by intravenous injection, usually in a dose of 0.48 gm. in 20 cc. of solution. In certain patients this drug relieves the substernal pain almost immediately, but if it does not do so, it should be followed promptly by the hypodermic administration of morphine sulfate. The dose of this drug is usually ¼ grain, but whenever the pain is exceptionally severe one should not hesitate to administer ½ grain. Subsequent doses of ¼ grain should be given at intervals of approximately one-half hour if the distress continues unabated. Immediately after the administration of aminophyllin, and regardless of whether morphine sulfate is necessary, the patient should be given atropine sulfate by hypodermic injection. LeRoy and Snider[1] have demonstrated that sudden death after experimental ligation of a coronary artery is due to ventricular fibrillation, and that this arrhythmia is caused by vagal coronary constriction which, in turn, results in generalized myocardial ischemia. If the vagal vasoconstriction is reduced or abolished by adequate amounts of atropine,

the incidence of sudden death after coronary artery ligation is considerably reduced. In man the usual initial dose of atropine sulfate is 1/75th grain, and additional doses of 1/150th grain are given every six or eight hours during the first three or four days.

The patient should be placed in bed as soon as possible after the onset of symptoms and should not be disturbed by frequent examinations. Because of the shock and profuse perspiration which often are present, the body should be kept warm, and as soon as the patient is more comfortable and is free from nausea and vomiting, small amounts of fluids should be offered at frequent intervals. Stimulants, such as caffeine with sodium benzoate, are administered only if the systolic blood pressure falls below 80 mm. of mercury.

In the more severe attacks of myocardial infarction which are accompanied by cyanosis and intense dyspnea, oxygen should be administered as promptly as possible, either by means of an oxygen tent, nasal catheter, or a B. L. B. mask. This measure not only reduces the cyanosis and dyspnea, but also may lessen the intensity and shorten the duration of the pain.

After the pain and initial shock have been controlled, most patients require little medication. Atropine should be continued in the prescribed dosage, and it probably is best to administer adequate amounts of one of the xanthine preparations, preferably theobromine with sodium acetate in doses of 7½ grains four times a day. If the heart rate remains unduly elevated or if frequent premature beats develop, quinidine sulfate should be given in doses of 3 grains or 5 grains every three or four hours as a possible prophylactic measure against the development of ventricular paroxysmal tachycardia and ventricular fibrillation. Sedatives may be necessary in small divided doses during the day or at bedtime to control restlessness. Digitalis is administered only in the event of congestive myocardial failure, or when there is auricular fibrillation with a rapid ventricular rate. The diet should be simple and should be limited to a value of 800 or 1000 calories. If the bowels do not move spontaneously, enemas should not be administered until after the second or third day. The emphasis in treatment should be placed upon the necessity for absolute rest. The patient should be fed and should not be allowed to help in changing his position in bed for at least three weeks. The total period of rest in bed should be from six to eight weeks. The erythrocyte sedimentation rate is a helpful guide in this respect; rest is enforced until the rate shows a considerable return toward normal and reaches a stationary level. After the period of rest, the patient is permitted to be up for gradually increasing lengths of time daily, but is not allowed to return to his business activities for from three to twelve months, depending upon the severity of the attack.

ADVANCED CONGESTIVE HEART FAILURE

The second most common type of cardiac emergency is encountered in patients who have congestive heart failure and are first seen after their condition has become critical. In situations of this kind the patient usually has experienced increasingly severe symptoms for days or even weeks, and examination reveals an exhausted, apprehensive individual in extreme respiratory discomfort and often in a condition bordering upon shock. Orthopnea is present, and there is cyanosis, engorgement of the jugular veins, an enlarged, tender liver, and extensive peripheral edema. Hydrothorax and ascites may be present, and in many patients there is repeated vomiting. The cardiac rhythm may be regular or irregular; the most common type of arrhythmia observed is auricular fibrillation with a very rapid ventricular rate and a large radial pulse deficit.

In an emergency of this kind the first indication is for the administration of digitalis, and because a delay of even a few hours in obtaining the therapeutic effect of the drug may mean the difference between a fatal and a successful outcome, intravenous administration is necessary. It is important to remember in this connection that when digitalis action is urgently needed, one must not rely upon intramuscular injection. There are several preparations of digitalis which are marketed in ampules for intravenous use. Unfortunately, these preparations differ considerably in potency among themselves, and to a somewhat lesser extent, in different lots of the same product. It is therefore advisable that one become familiar with the clinical action of a single preparation and confine himself to the use of that product. For several of the preparations on the market, an initial intravenous dose of 10 cc. is suitable, and the same amount may be given again if necessary after an interval of four hours. After this, it is generally advisable to complete the process of digitalization either by oral administration of the drug, if vomiting has ceased, or by intramuscular injection.

Strophanthin may be given intravenously in place of one of the digitalis preparations. This drug, however, has no definite advantage over digitalis and is dangerous when given in large doses. The initial dose should be not more than 0.5 mg. Additional doses of 0.1 mg. may be given if necessary at intervals of four hours until a total of not more than 1 mg. has been administered. It is, of course, important to ascertain that patients to whom digitalis or strophanthin is given intravenously have not received digitalis during the preceding two weeks.

The administration of digitalis or strophanthin may produce remarkably prompt improvement in the patient who is critically ill with congestive heart failure, particularly when auricular fibrillation is present. In patients with auricular fibrillation, slowing of the ventricular

rate usually is noted within five minutes after administration of the drug, and within thirty minutes there may be complete cessation of vomiting and great diminution in the degree of dyspnea. Strophanthin attains its maximum effect in approximately one hour and the digitalis preparations in about two hours after intravenous injection.

In addition to receiving digitalis or strophanthin intravenously, patients who are desperately ill with congestive failure should be given morphine sulfate promptly by hypodermic injection. This drug depresses the respiratory and higher cerebral centers and relieves the patient's dyspnea and his anxiety and apprehension. Not only should morphine be administered when the patient is first seen, but a second injection, usually of ¼ grain, also should be given the same evening to insure a comfortable night's rest. Several hours' sleep frequently results in striking improvement in the general condition and morale of the patient.

Advanced congestive failure often is attended by the accumulation of large amounts of fluid in the serous cavities of the body. Extensive hydrothorax may be present on one or both sides and, by compressing the lung, may be responsible for a considerable part of the reduction in vital capacity and resultant dyspnea. It is important, therefore, that the fluid be removed as completely as possible soon after the initial administration of digitalis and morphine. Less frequently, ascites is present in sufficient amounts to interfere with the movement of the diaphragm and to contribute to the degree of dyspnea. Under such circumstances, abdominal paracentesis is indicated.

In patients with myocardial failure, the peripheral venous pressure is increased approximately in proportion to the degree of decompensation. In severe failure the jugular veins may be engorged to the angle of the jaw even with the patient well propped up in bed. Venesection with the removal of 350 to 600 cc. of blood may result in prompt improvement in cases of this kind and should be employed whenever the institution of digitalis therapy and the other measures mentioned previously fail to produce satisfactory improvement. This procedure directly reduces venous congestion and diminishes the degree of dilatation of the heart. It is desirable to measure the venous pressure during the removal of the blood and to continue the bleeding until the pressure has been reduced to within the upper limits of normal. For practical purposes, however, a reliable guide is furnished by observation of the jugular veins, venesection being continued until jugular distention has been relieved. In favorable cases the venous pressure remains low after having been reduced by venesection, but in unfavorable cases peripheral venous congestion promptly returns.

CARDIAC ASTHMA AND ACUTE PULMONARY EDEMA

Cardiac asthma is a form of paroxysmal dyspnea which occurs in patients who have serious organic heart disease. Its onset occasionally gives the first warning of a damaged heart, but more often the patient has experienced dyspnea or anginal pain on effort for some time before the first seizure. The attacks develop rapidly and are characterized by asthmatic breathing with both inspiratory and expiratory difficulty, orthopnea, and a sense of suffocation. The paroxysms may last from several minutes to a few hours, and death may occur during the attack.

In all but a few cases cardiac asthma is due to failure of a left ventricle which has been damaged previously as the result of hypertension, coronary artery sclerosis, or aortic valve disease. The seizures in these cases usually occur at night, although occasionally they are induced by exertion. Because of relative weakness of the left ventricle, an increased amount of blood gradually accumulates in the pulmonary vessels during sleep in the recumbent position. The vital capacity, which is already diminished, is further reduced as the degree of pulmonary congestion increases, and all that is now needed to initiate the attack of cardiac asthma is some factor which acts as a trigger mechanism. Cough, Cheyne-Stokes respiration, noise, disturbing dreams, and the urinary reflex most commonly supply this factor. The patient wakens with respiratory distress and is forced to sit up or stand in order to breathe. Asthmatic breathing develops, and as the attack progresses, acute pulmonary edema may supervene.

In a much smaller group of patients cardiac asthma results from advanced mitral stenosis without myocardial failure. A series of such cases has been studied by McGinn and White[2] who point out that, in contrast to attacks resulting from failure of the left ventricle, the seizures in uncomplicated mitral stenosis usually are precipitated by exertion, emotional upsets, or paroxysmal tachycardia. When the heart rate is accelerated by any of these factors, the hypertrophied right ventricle expels blood into the pulmonary circulation more rapidly than it can pass through the narrowed mitral orifice. Acute pulmonary congestion develops and produces a paroxysm of cardiac asthma.

The most important measure in the treatment of the attack of cardiac asthma due to left ventricular failure is the intravenous administration of aminophyllin, usually in a dose of 0.48 gm. in 20 cc. of solution. This may produce prompt and lasting improvement. The beneficial effect of the preparation has been attributed principally to its action on the coronary circulation,[3] but the drug also causes a diminution in the degree of bronchial spasm.[4] It is known that bronchial spasm is present during the paroxysm of cardiac asthma and undoubtedly contributes importantly to the degree of dyspnea. It may

be, therefore, that the beneficial effect of aminophyllin is due as much to its effect on the bronchial musculature as to its action on the coronary arteries. If the drug does not prove sufficiently beneficial, it should be followed promptly by the hypodermic administration of morphine sulfate, ¼ grain, which should be repeated if the patient is not improved within 15 or 20 minutes. Venesection, with the removal of 350 to 500 cc. of blood should be carried out in patients in whom the venous pressure is elevated and may prove to be a very helpful measure. An effect similar to that of venesection may be obtained by applying blood pressure cuffs to the four extremities and inflating them to a pressure just above diastolic blood pressure. The administration of oxygen by means of a tent, mask, or nasal catheter is also of great value and should be instituted whenever aminophyllin and morphine do not give sufficient relief.

When cardiac asthma progresses to acute pulmonary edema in spite of the above measures, either strophanthin or a suitable preparation of digitalis should be given by intravenous injection. It is, of course, essential to ascertain that these patients have not received digitalis previously.

The treatment of cardiac asthma due to uncomplicated mitral stenosis is similar to that employed for attacks resulting from left ventricular failure. When the seizures are due to paroxysmal auricular fibrillation, rapid digitalization should be carried out, and when the precipitating factor is paroxysmal tachycardia, suitable measures for this condition must be employed.

A patient who has experienced an attack of cardiac asthma due to failure of the left ventricle should be treated as any other individual who presents evidence of impaired myocardial reserve. Complete digitalization and the subsequent administration of daily maintenance doses of the drug are indicated in individuals who have had but mild attacks, and this may suffice to prevent the recurrence of paroxysms. In those who have suffered more severe attacks, a period of absolute rest is advisable and should be followed by strict limitation of activity. Restriction of fluids, a diet low in sodium chloride content, and the administration of diuretic drugs also are valuable measures. At times, the intravenous injection of hypertonic glucose solution (50 cc. of a 50 per cent solution daily for several days) helps to reduce the frequency of the attacks. Because cardiac asthma due to left ventricular failure usually occurs at night and because the onset of the seizure is favored by the recumbent position, the patient should be instructed to sleep well propped up in bed. Sedatives should be used with caution since their depressant effect upon the respiratory center may favor rather than hinder the development of attacks.

In cardiac asthma due to uncomplicated mitral stenosis, digitalis is seldom effective in preventing the recurrence of attacks except when the paroxysms are induced by auricular paroxysmal tachycardia. Diuretics also are of little value, and therefore it is of great importance that these patients avoid exertion and emotional upsets which may precipitate an attack. Sedatives should be given daily in divided doses to those patients who display evidence of emotional instability.

ACUTE COR PULMONALE

Sudden embolic occlusion of the pulmonary artery or its primary branches may be immediately fatal or may cause severe dyspnea associated with substernal oppression and the rapid development of a state of shock. If the patient survives the onset of the attack, evidence of prompt dilatation and failure of the chambers of the right side of the heart develops. McGinn and White[5] have termed this cardiac disturbance "the acute cor pulmonale." They reported nine cases of acute cor pulmonale and described the clinical and electrocardiographic features which differentiate the condition from acute myocardial infarction. The most important clinical features are an increased pulsation palpable in the left second intercostal space adjacent to the sternum, accentuation of the pulmonary second sound, the frequent occurrence of gallop rhythm over the pulmonary area, the occasional presence of a friction rub in the second and third interspaces adjacent to the sternum, and, unless the patient is in deep shock, engorged and distended neck veins in the absence of signs of passive congestion in the lung bases.

The medical measures employed in the treatment of acute cor pulmonale due to pulmonary embolism consist of the administration of oxygen by tent, mask, or nasal catheter, and the intravenous administration of papaverine hydrochloride, ¼ grain, or atropine sulfate 1/100 grain. Morphine may be necessary for the control of pleural pain. Surgical removal of the embolus has been attempted, and in rare instances the operation has been successful.

DISTURBANCES OF CARDIAC RHYTHM

Certain disturbances of cardiac rhythm are sufficiently important to be classified as cardiac emergencies, while others are of little significance, although they may produce symptoms that alarm the patient. Ventricular paroxysmal tachycardia is an example of the first type of disturbance, and auricular paroxysmal tachycardia belongs to the second group. Ventricular paroxysmal tachycardia is a relatively uncommon condition which usually is due to serious organic heart disease. It occurs most commonly as a complication of myocardial infarction and may be a forerunner of ventricular fibrillation. Occasionally, however, it is encountered in the absence of any evidence of organic heart disease. In

patients who have coronary artery disease, the paroxysm of tachycardia may be attended by serious collapse and occasionally by the rapid development of acute pulmonary edema. Levine[6] has pointed out that in many cases ventricular paroxysmal tachycardia may be recognized by clinical means alone. Carotid sinus or ocular pressure and breath-holding do not affect the heart rate in this condition as they do in auricular paroxysmal tachycardia and auricular flutter. Furthermore, if one listens carefully to the heart, an occasional slight irregularity in rhythm will be noted, and the intensity of the first heart sound will vary from time to time. The one drug of established value in the treatment of this type of tachycardia is quinidine sulfate administered by mouth. Usually an initial dose of 3 grains is given, and this is followed at intervals of two hours by additional doses of 6 grains each until sinus rhythm is reestablished. Morphine may be necessary to relieve dyspnea, and the occurrence of acute pulmonary edema calls for the administration of oxygen.

Paroxysmal auricular flutter seldom occurs in the absence of organic heart disease. When the ventricular rate is elevated to 160 beats per minute or more, the condition may be responsible for the rapid development of congestive heart failure. In those cases in which the ventricular rhythm is regular and the rate between 120 and 200 beats per minute, the disturbance must be differentiated from sinus tachycardia and from auricular paroxysmal tachycardia. The electrocardiogram affords the most precise means of establishing the diagnosis, but the arrhythmia can often be recognized without instrumental aid. Careful inspection of the venous pulsations in the neck may clearly reveal two or more auricular pulsations to each ventricular wave. In auricular flutter the ventricular rate remains constant within very narrow limits and is not appreciably affected by exercise. This is in contrast to the variability of the rate in sinus tachycardia, but does not aid in distinguishing the condition from auricular paroxysmal tachycardia. Pressure upon the carotid sinus may cause temporary slowing of the ventricular rate in auricular flutter, or it may produce an abrupt standstill of the heart for a variable length of time, followed by resumption of the original rate. The first of these responses may be obtained in patients with sinus tachycardia, but the second does not occur. In auricular paroxysmal tachycardia pressure upon the carotid sinus either produces an abrupt reversion to normal rhythm, or has no effect at all. The ventricular rhythm in auricular flutter may be irregular because of variations in the degree of auriculoventricular block, and at times the irregularity is sufficiently marked to suggest auricular fibrillation. Careful auscultation usually reveals an underlying dominant rhythm, however, and this distinguishes the condition from auricular fibrillation.

Although the administration of quinidine sulfate may convert auricular flutter to sinus rhythm, the preferred treatment for the arrhythmia consists of rapid digitalization. In successful cases digitalis converts auricular flutter to auricular fibrillation, and discontinuance of the drug may then be followed by spontaneous resumption of normal sinus rhythm.

Auricular paroxysmal tachycardia is encountered much more often in individuals who have normal hearts than in those who present evidence of organic heart disease. Although the patient may be greatly disturbed by weakness, light-headedness, and palpitation incident to the attack, the tachycardia seldom produces evidence of coronary or myocardial insufficiency. Occasionally, however, in the presence of organic heart disease, the patient may experience severe substernal pain or develop symptoms and signs of congestive myocardial failure. The paroxysms usually begin and terminate suddenly and are characterized by a perfectly regular rhythm, generally with a rate between 160 and 200 beats per minute. There are a number of simple procedures that often are effective in abruptly terminating the attack. The most reliable of these are carotid sinus or ocular pressure and breath-holding. When these measures fail, drug therapy should be employed, and the most effective preparation is mecholyl by subcutaneous injection. The usual initial dose of this drug for adults is 20 mg., and a second dose of 20 mg. may be given, if necessary, after 20 or 30 minutes. In many cases normal heart rhythm will be reestablished within 15 minutes after the first injection, and before administering a second dose, it is best to massage the site of the injection and repeat the carotid sinus or ocular pressure, or breath-holding. A syringe containing atropine sulfate, 1/100 grain, should be prepared before the first dose is administered, so that this drug can be given by intravenous injection if mecholyl causes disagreeable or alarming symptoms of excessive parasympathetic stimulation. Mecholyl should not be employed in individuals who have bronchial asthma or coronary artery disease. If the drug does not control the tachycardia, quinidine sulfate may be given by mouth in the manner outlined for the treatment of ventricular paroxysmal tachycardia. If this also fails, moderately rapid digitalization should be instituted and may be effective in terminating the tachycardia.

The higher grades of auriculoventricular block, and particularly complete auriculoventricular dissociation, may be complicated by Adams-Stokes attacks due to temporary standstill of the ventricles. The seizures are characterized by dizziness, syncope, or convulsions, depending upon the duration of the ventricular asystole. Adams-Stokes attacks are not common, but individuals in whom they occur are liable to have repeated seizures. The actual attacks are of such short duration that

they seldom require treatment, and therapy therefore is directed toward preventing their recurrence. Occasionally, however, the standstill of the ventricles is of such duration as to necessitate the intracardiac injection of epinephrine, and this procedure may be directly responsible for the saving of life. The most effective drugs for preventing recurrent attacks are epinephrine (0.3 cc. to 1.0 cc. of the 1:1000 solution) by intramuscular injection every three or four hours and ephedrine sulfate (⅜ grain to ½ grain) by mouth three or four times in 24 hours.

ACUTE CARDIAC COMPRESSION

Acute compression of the heart results from the rapid accumulation of serous exudate, pus, or blood in the pericardial sac. In the usual case of serous or sero-fibrinous pericardial effusion, the amount of fluid present is not sufficiently large to embarrass the circulation importantly, and pericardial paracentesis therefore is not necessary. Occasionally, however, an effusion becomes so extensive that the superior and inferior vena cavae are compressed and the return flow of blood to the right auricle is interfered with. Unless this excessive intrapericardial pressure is relieved, the condition will rapidly prove fatal. The most important indications that a pericardial effusion is attaining dangerous proportions are orthopnea, cyanosis, greatly elevated venous pressure, and a rapid fall in arterial blood pressure with a small pulse pressure. The development of these symptoms and signs indicates the need for paracentesis with the removal of as much fluid as possible without producing pain, cough, or faintness. The procedure is carried out under novocaine anesthesia, and the needle usually is introduced either in the left fifth intercostal space just within the outer border of dulness, or in the right fourth intercostal space just inside the right border of dulness.

Purulent pericarditis is an unusual condition which generally occurs as a complication of pneumococcus pneumonia or staphylococcus osteomyelitis. The development of signs of pericardial effusion and acute compression of the heart in these conditions should be interpreted as an indication for immediate paracentesis. The discovery that the effusion is purulent calls, in turn, for surgical drainage.

Hemopericardium with resultant acute cardiac tamponade may result from a number of conditions, but the only form amenable to treatment is that due to penetrating wounds of the heart. The treatment is, of course, surgical, but a successful operation depends directly upon prompt recognition of the condition.

SUMMARY

The most frequently encountered cardiac emergencies are acute myocardial infarction, far advanced congestive heart failure, cardiac

asthma, and certain disturbances of heart rhythm. Less common emergencies consist of acute compression of the heart due to the rapid accumulation of blood, pus, and serous exudate in the pericardium, and acute cor pulmonale. The diagnosis and treatment of each of these conditions have been discussed.

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THE PRESENT STATUS OF CASTRATION FOR CARCINOMA OF THE PROSTATE

C. C. HIGGINS, M. D. and C. L. GOSSE, M. D.

The treatment of carcinoma of the prostate has long been considered a major problem in the practice of urology. Resection of the gland by one technic or another, x-ray therapy, or both have been the chief methods of treatment. However, the condition has been so far advanced when the patient has presented himself that the outlook has been pessimistic. Now and then hope has been offered by some new therapeutic measure, but the proof of time and experience usually has been lacking.

Today castration as a new method of treatment seems to promise gratifying results. This procedure may be compared with oophorectomy for carcinoma of the breast in that both are on a hormonal basis. However, from early conservative reports castration for prostatic carcinoma seems to have a definitely more favorable outlook.

It has been shown that when androgenic hormones are reduced sufficiently, prostatic epithelium undergoes atrophy. Conversely, the injection of adrogens stimulates the growth of prostatic epithelium, and the injection of estrogens retards its growth. Furthermore, eunuchs who are without a primary source of androgens do not develop prostatic hypertrophy.

Carcinoma of the prostate results from overgrowth and invasion of adult epithelial cells. Concrete evidence of this has been shown by phosphatase estimations. Gutman and Gutman[1, 2] and Kutscher and Wolbergs[3] made extensive studies of the subject. The former examined prostate glands at various ages for acid phosphatase. They observed low values in children, a slight increase through puberty, and high values in adults. The phosphatase values for carcinoma of the prostate were similar to those for the adult gland. Huggins et al,[4, 5, 6] confirmed this work by staining methods and found that the enzyme phosphatase of normal adult and hypertrophied prostates compared in quantity with that of carcinomatous glands. From this they concluded that the prostatic carcinomas examined consisted of adult rather than of more primitive neoplastic epithelium. Further evidence has been found in the levels of urinary keto-steroids. Slatterthwaite, Hill, and Packard[7] showed that the mean level of excretion of 17 keto-steroids in ten cases of benign prostatic hypertrophy did not vary significantly from that in carcinoma of the prostate.

The determination of the serum phosphatase is a valuable aid in the diagnosis of metastatic bone lesions from carcinoma of the prostate. It also helps to evaluate the effects of therapy and may be significant in determining the patient's prognosis.

The enzyme phosphatase has been found in body tissues and fluids in varying amounts. It has the ability to liberate phosphate ions from a solution containing esters of phosphoric acid. Two methods of determination are the King-Armstrong and the Bodansky. Both express the phosphatase activity in units. The pH of the medium determines the amount of phosphatase activity. The optimum pH for acid phosphatase is 4.9, whereas that for alkaline phosphatase is 8.6. One acid phosphatase unit (K-A) is defined as "that degree of phosphatase activity which at pH 4.9 and 37 degrees C. will liberate 1 mg. of phenol from the specific buffer—monophenylphosphate substrate solution—in one hour. The number of units is determined to express the amount of phosphatase activity in 100 cc. of blood serum."[5]

The acid phosphatase level is undoubtedly more significant in cases of carcinoma of the prostate, and whenever it has been increased appreciably, metastasis has invariably been present.[8] However, metastasis has also been present when the acid phosphatase levels have been normal. Thus there are false negative but no false positive reactions. The normal level is from 0 to 4 K-A units, although 4-6 units is considered high normal under certain circumstances.

The alkaline phosphatase level may be considered as a diagnostic adjunct to acid phosphatase in borderline cases. The normal is from 0 to 6 units. Herger and Sauer[8] warn that in the interpretation of 4-6 units of acid phosphatase activity the utmost caution should be exercised when the alkaline phosphatase is under the average normal of 6 units or elevated above 12 units, and that frequent determinations should be made. Alkaline phosphatase estimations are also of value in the differential diagnosis of bone metastasis from carcinoma of the prostate and Paget's disease of the bone.[10] In the latter the acid phosphatase level is usually normal, whereas the alkaline phosphatase is increased. High alkaline phosphatase readings are also obtained in active rickets, generalized osteoporosis, hyperthyroidism, and biliary obstruction. In these conditions acid phosphatase levels are usually normal.

Castration for carcinoma of the prostate has been performed by Huggins et al,[5] and their series of 21 cases has been reported in detail. Nine cases showed osteoplastic metastasis, six showed osteolytic, and the remaining six cases presented no evidence of metastasis. The average age of the patients was 69, the youngest being 54, and the oldest 84 years of age.

In the last 14 cases serum phosphatase levels were recorded before and after orchidectomy. Phosphatase levels associated with the various metastatic bone lesions were within the following limits:

Phosphatase Levels in K-A Units

Nature of Metastasis	Acid Phosphatase	Alkaline Phosphatase
Osteoplastic	22 — 75 units	22— 78 units
Osteolytic	1.5— 37 units	6— 24 units
None	1 —4.5 units	6—9.75 units

Castration was performed under local anesthesia in most cases. Transurethral resection of the prostate was performed in seven instances, and suprapubic prostatectomy in one case. Roentgen therapy was not used in any of the patients. Pathologically, the testis revealed nothing of significance.

Three patients in this series died within a few months of cardiac disease or pneumonia. Another patient died in 234 days without any pronounced relief of symptoms or other improvement. The remaining patients maintained gains in weight of 3 to 18 kg. within two to 18 months. The trend of the erythrocyte count was upward. Pain which had been prominent in patients with metastasis showed either a recession or complete remission within two to eight weeks after castration. Of the seven patients previously confined to bed, six became ambulatory. One patient with involvement of the cauda equina showed resolution of the lesion. Sexual desire and penile erections were absent, but no mental changes were noted. Hot flashes similar to those experienced by women after the menopause were evident in six cases, but were relieved by the use of stilbestrol in small doses.

By rectal palpation the prostate gland was found to be diminished in size in the majority of cases to the extent of being just preceptible or actually impalpable within 12 weeks. This decrease in size was maintained throughout the periods of observation, the longest of which was 18 months. Roentgenograms taken following operation showed increased osteosclerosis within three to six months. Whether or not this phenomenon signifies remission is still a matter of conjecture. High acid phosphatase levels before castration usually became or approached normal following operation. When the normal was not attained, it was believed that there may have been an increased extratesticular output of androgens.

Satterthwaite, Hill, and Packard[7] have reported a series of ten cases of extensive carcinoma of the prostate in which castration was performed. These workers made a study of the excretion of the 17 ketosteroids in the urine both preoperatively and postoperatively as well as

phosphatase levels and found a general reduction in the output of the 17 keto-steroids, varying from 12 to 60 per cent of the preoperative level.

Their results seem to be almost as promising as those of Huggins et al.[5] Two patients had "complete relief from pain, increase in appetite, gain in strength, a marked decrease in obstructive symptoms, and a drop in the basic phosphatase to normal levels." These two cases which showed the most improvement had a drop in the 17 keto-steroid output; in one case from 2.3 to 0.9 and in the other from 12.6 to 6.7 mg. in 24 hours. One patient who showed virtually no improvement had a reduction of from 3.9 to only 3.4 mg. in 24 hours. In the other seven cases clinical improvement apparently coincided with the reduction in the urinary output of the 17 keto-steroids. These workers suggest the advisability of estrogen therapy as an adjunct in cases in which sufficient reduction of 17 keto-steroids is not obtained.

Simple bilateral orchidectomy can hardly be considered a major operation. However, it requires at least two or three days' bed rest and is associated with a certain amount of discomfort following the procedure. Consequently, irradiation to the testicles as a substitute for orchidectomy is worthy of consideration. Only a small amount of irradiation is necessary to destroy the spermatogenic cells of the testicle. However, the interstitial cells which are responsible for the androgen supply are comparatively radioresistant. Munger[9], however, has shown clinically that supervoltage irradiation of the testicle has produced diminution of the symptoms and physical findings and comparatively has increased longevity. His measurements were purely qualitative, and little quantitative laboratory or other data were recorded. He reported 76 cases of carcinoma of the prostate in patients admitted to his service in a five year period ending December 1938. At the time of the report (May 1941) 51 patients were dead, seven were unaccounted for, and 18 were alive. Of the 76 patients 12 had prostatic resection alone; 45 had resection and irradiation to the prostatic area; and 11 had resection, irradiation, and an additional 500 r supervoltage therapy to the testicles.

The results were rather significant. Of the 12 patients which had resection alone, all died. Of the 45 who had resection and irradiation, only ten were alive. Of the 11 who had resection, irradiation, and adjunct x-ray therapy, eight were still alive. Furthermore, this latter group had improved symptomatically, and the prostatic bed by rectal examination was found to be "smooth, resilient, and free of nodules." The longest period of survival was seven years and the shortest three years.

A comparison of the results from irradiation of the testicles with the results from orchidectomy is difficult to establish. The scientific data are not quantitatively available from the cases in which irradiation

of the testicles was performed. The keto-steroid and phosphatase levels were not recorded. Therefore, a common basis for accurate judgment obviously is lacking. Consequently, more detailed studies must be made before drawing any definite conclusion.

No one can deny castration a scientific parentage, nor that, according to early reports, the procedure is warranted, especially since there is much to gain and little to lose. Even if only the intense pain from bony metastasis has been alleviated and the patients allowed to die in comparative comfort, something has been accomplished. The scientific material at hand is by no means sufficient to warrant its adoption without reservation as to the end results. In the presence of obstruction transurethral resection also may be advisable.

The Cleveland Clinic has followed the procedure of advising orchidectomy for carcinoma of the prostate, or orchidectomy and transurethral resection for nearly a year. The results have been variable, but on the whole have been successful enough to justify the procedure.

Results: Our series consisted of 16 cases. The average age of the patients was 69 years. In 12 of the 16 cases there was no evidence of metastases, while four showed an osteoplastic type of lesion chiefly of the pelvis and spine which was observed on x-ray examination. In cases without metastases the blood counts for the most part were normal; those with metastases showed varying degrees of anemia. The acid phosphatase ranged in the nonmetastatic cases from 1.0 to 2.6 units and in cases with metastases from 5.6 to 80 units.

Nine of the 16 cases had transurethral resections of the prostate as well as orchidectomy. In seven cases there was no biopsy of the prostate. The nine cases in which biopsy was obtained at the time of transurethral resection showed four adenocarcinomas, three undifferentiated carcinomas, one medullary duct type of carcinoma, and one papillary carcinoma. It was noted that bony metastasis was present only in those cases which showed adenocarcinomatous growths.

In five instances only a few weeks had elapsed since operation, or no follow-up data were obtained. Seven cases showed definite improvement in relief from symptoms. One showed a gain in weight but developed slight incontinence for some unexplained reason (transurethral resection was not performed); and three showed an increase in appetite and a gain in weight, diminution in the size of the gland by rectal examination, diminution in the obstructive symptoms, and lessening or complete remission of pain. Furthermore, these patients felt improved generally.

In four cases subsequent laboratory estimations were made, and in these the acid phosphatase, which originally had been normal, showed a decrease varying from 0.1 unit to 1.8 units over periods ranging up to five

months. In two cases subsequent alkaline phosphatase estimations were made, both of which had showed normal readings. In one case the change was from 2.5 units on the day of operation to 2.8 units four months later. In the other case the reading was 1.9 units at operation and 1.7 units six weeks later. In two cases 17 keto-steroid estimations were made preoperatively and postoperatively. Before operation the first case showed an output of 3.3 mg. per 24 hours; 8.8 mg. the day following operation; and 5.6 mg. six weeks later. The second case showed an output of 7 mg. per 24 hours before operation and 4.3 mg. two days after operation. In another case the 17 keto-steroid estimation one month following operation was 4.6 mg. per 24 hours. Two months postoperatively it was 7.5 mg.

Only when there had been definite anemia was the trend of the erythrocyte count decidedly upward. Hot flashes occurred in three cases, but there were no mental changes. Penile erections and sexual desire were absent in the majority of cases postoperatively.

CASE REPORT

The following case report is illustrative.

The patient was admitted on June 19, 1941 with complaints of frequency, urgency, dysuria, dribbling, and nocturia as many as fifteen times. There was considerable pain in the lumbar region, pelvis, and thighs. These symptoms had been of approximately six months' duration. During this time he had lost 25 pounds in weight. There was no history of hematuria.

Upon rectal examination the prostate gland revealed grade IV enlargement, was hard, nodular, and fixed, and there was marked extension to both vesicles. The residual urine was 285 cc. The blood count upon admission showed 4,980,000 red blood cells, 91 per cent hemoglobin, and 5,500 white blood cells. The urine and blood chemistry studies were essentially normal. Estimation of alkaline phosphatase was 2.5 units per 100 cc. serum; acid phosphatase 1.8 units per 100 cc. serum. There was no evidence of bony metastasis by x-ray.

On June 27, 1941, under low spinal anesthesia, a bilateral orchidectomy was performed. The patient was discharged from the hospital on the tenth postoperative day after an uneventful convalescence.

Subsequent acid phosphatase estimations were as follows:

6/28/41	2.1	8/19/41	3.0
6/30/41	1.9	10/31/41	1.1
7/1/41	1.9		

Alkaline phosphatase estimations were:

6/28/41	2.7	7/2/41	2.6
6/30/41	2.7	8/19/41	3.0
7/1/41	2.6	10/31/41	2.8

17 keto-steroid estimations were:

6/30/41	11.3 mg. per 24 hour specimen
11/4/41	13.0 mg. per 24 hour specimen

Follow-up examination on February 23, 1942 showed the patient to be generally improved in appearance. His symptoms had entirely disappeared; pain was absent; there was marked improvement in the caliber of the urinary stream; and no residual urine was noted. He had gained 50 pounds in weight since the operation. The gland

of the testicles was performed. The keto-steroid and phosphatase levels were not recorded. Therefore, a common basis for accurate judgment obviously is lacking. Consequently, more detailed studies must be made before drawing any definite conclusion.

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had diminished in size to almost normal; there were no palpable nodules; and the consistency of the gland had changed from stony hardness to a more normal one. However, the extension to the vesicles seemed to be just as prominent if not more so. Sexual desire was absent, and penile erections were impossible. However, there were no mental changes. The patient had been having hot flashes once daily which lasted for about five minutes and were associated with flushing and perspiration.

SUMMARY

Orchidectomy appears to be a valuable adjunct in the treatment of carcinoma of the prostate.

Individualization of the patient is essential, as in some cases transurethral resection is also required to relieve the immediate obstruction. However, not until a large number of cases have been so treated, and detailed laboratory and clinical data compiled over a longer period of time, will we be able to evaluate scientifically the results of this procedure.

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CHRONIC ENCEPHALITIS

A 20 Year Clinical Study — Case Report

JOHN TUCKER, M.D.

In major epidemics of encephalitis lethargica it is estimated that about 25 per cent of the patients recover completely without sequelae.[1] Of the remainder some die, and the others develop neurological disorders of one sort or another. Not all of these patients show progressive degenerative changes in the striatal and parastriatal nuclei of the midbrain to the extent that severe parkinsonism develops. Furthermore, when one considers the diffuse damage to the gray matter of the brain, it is not surprising that certain patients develop bizarre disturbances of the sensory and motor functions of the body.

If the symptoms of the acute onset are characteristic of the lethargic or epidemic type of encephalitis, the neurological changes may be attributed to this disease with reasonable certainty. On the other hand, even though great care is used in the clinical study of any particular case, a long period of observation may be required before a proper diagnosis can be made. The following case is of a patient whose symptoms and physical signs were puzzling and who at first seemed to be headed for postencephalitic parkinsonism.

CASE REPORT

This case of chronic encephalitis is of considerable interest not only because it has been followed clinically for 20 years during which time the patient has married, raised three children, and has earned his living; but also because he has not showed the usual progressive changes in the basal ganglia and hypothalamic areas of the brain. Another important feature of this case has been the recent occurrence of spells of unconsciousness which have resembled the grand mal seizures of idiopathic epilepsy. Furthermore, the clinical differentiation between encephalitis, encephalomyelitis, and disseminated sclerosis has presented an interesting and difficult diagnostic problem.

A young man, 22 years of age, was admitted to the Clinic in September, 1922. His chief complaints were marked generalized fatigue, dizziness, fullness in the head, difficulty with speech, and moderate ataxia. In February, 1920 he had had an attack of "influenza" characterized by moderate fever, malaise, and muscular aching. He had recovered from the acute illness within a few days, but had complained of continued fatigue until July, 1920. At that time symptoms of encephalitis had developed with diplopia, somnolence, and dizziness. His speech became disturbed by difficulty in use of the tongue, although swallowing was not affected. He improved in the next two months, but he continued to experience moderate ataxia, dysarthria, and excessive salivation.

At the initial examination at the Clinic in September, 1922 the dysarthria was still present as well as dizziness, moderate ataxia, generalized fatigue, and marked constipation.

Neurological Study: An examination of the eyes revealed that the optic discs were normal. The pupils were equal and regular and reacted to light and convergence. There was no ocular palsy. Nystagmus was present on looking to the right.

The patient had a moderate mask-like expression with infrequent winking. Weakness was especially marked on the left side of the face when in repose, and there was hypesthesia of the left face.

The reflexes of the upper extremities were normal. In the lower extremities there were hyperactive patellar reflexes and a positive Babinski on the right.

The abdominal reflexes were present but sluggish; the cremasteric reflexes were also present.

The muscles of the right arm and leg were slightly spastic. There was no atrophy nor tremor. The finger to nose and heel to knee test was awkward on the right. The patient's gait was slightly ataxic from spasticity of the right leg.

Laboratory Studies: The blood Wassermann and Kahn reactions were negative. Blood counts revealed no macrocytosis. An Ewald test meal revealed a free acid of 42 and a total acid of 56 in one hour. The patient refused spinal puncture.

The clinical diagnosis was a diffuse lesion of the cortex and basal ganglia from acute encephalitis and probably early parkinsonism.

Treatment: With the use of Fowler's solution and potassium iodide the patient gained 21 pounds in two months and was able to return to work.

Progress Notes: In June, 1923, nine months later, the patient complained of numbness of the right side of the body and of marked numbness and hypesthesia of the palm of the right hand. His general condition was good.

In May, 1924 examination revealed numbness in the toes of both feet with diminution of sensation to touch, pin prick, and vibration.

In April, 1929 examination revealed numbness of the left side of face and the left half of hard palate and tongue, with loss of the sense of taste over this area. A speech difficulty was again present. There were hyperactive reflexes in the legs with bilateral ankle clonus and a right Babinski. Abnormal reflexes were absent, and there was no ataxia.

At this time the patient submitted to a spinal puncture. The subarachnoid fluid was clear and colorless, and the pressure was normal. Spinal fluid analysis revealed 2 cells; the globulin and Wassermann reactions were negative; colloidal gold, 3-2-2-1-1-1-0-0-0-0.

In October, 1937 the patient had numbness in the right leg, ataxia in the right leg on walking, and dysarthria.

In July, 1940 examination revealed a slight slurring of speech, a moderate intention tremor of right hand, weakness, and a sense of fatigue.

In 1938 the patient had a convulsion which was nocturnal and associated with urinary incontinence, but the tongue was not bitten.

In June, 1942 the second convulsion occurred. The patient felt himself pulled to the left and fell unconscious. When he recovered his senses after two hours, he was standing at his desk dressed in his overcoat and hat. No head injuries were noted, nor was the tongue bitten.

On February 5, 1942 at 9:30 p. m. while at home, he became unconscious and fell off of his chair. Convulsive movements involved the left arm and leg and lasted from five to six minutes. The inside of the right cheek was bitten. Upon recovery of consciousness within a few minutes, he experienced no pain nor headache, but felt dazed for an hour.

A neurological examination in February, 1942 revealed the following: There was no nystagmus nor ocular palsy. The pupils were equal and reacted normally; the optic discs were normal. There was slight weakness of the left side of face. The other cranial nerves were normal, and there was no pallor. Sensation to vibration was poor in the feet and absent in the toes. Reactions to pain, heat, and cold were normal.

Reflexes: The patellar and Achilles reflexes were hyperactive bilaterally. The Babinski reflex was present, the sustained ankle reflex was clonic on the right. The abdominal reflexes were active in the upper areas and sluggish in the lower. Cremasteric reflexes were absent. Ataxia was noted in the left finger to nose test. There was slight muscular hypertonus of the right leg. The Romberg was to the right and backwards. The patient's gait was disturbed as a result of spasticity of the right leg.

Upon spinal puncture the fluid was found to be clear and colorless; there were normal dynamics of fluid, 7 cells, a faint trace globulin, 45 mg. total protein; the Wassermann and Romberg colloidal gold reactions were negative. Electro-encephalography showed abnormal waves in the frontal area of the brain.

Discussion: Although the acute stage of encephalitis lethargica may show the characteristic signs of fever, somnolence, diplopia, and ocular palsies, postencephalitic sequelae often appear in the absence of such early disturbances. Likewise, postencephalitic or chronic encephalitic symptoms may be manifested by disabilities other than chronic parkinsonism or oculogyric crises. Changes in personality, emotional instability, and various sensory or vasomotor symptoms indicate that the lesions are diffuse, widespread, and usually progressive. Although the "virus" shows a specific affinity for the gray matter of the cortex and basal ganglia, the damage is by no means confined to these areas. Toomey[2] has emphasized the existence of many varieties of encephalitis and has drawn attention to their atypical manifestations.

Although the case reported illustrates certain rather bizarre and confusing nervous symptoms, the history clearly indicates an acute onset of encephalitis followed by more or less permanent disability. Even though the nervous system changes have been slowly progressive, the occurrence of remissions and exacerbations suggested that actually the patient might be suffering from chronic encephalomyelitis or from disseminated lesions of multiple sclerosis. However, although the spinal fluid showed a mildly positive colloidal gold reaction to the first test in April, 1929, the fluid obtained in February, 1942 was normal. Positive colloidal gold reactions have been reported in some cases of encephalitis, but are more common in disseminated sclerosis and cerebrospinal syphilis. Also, the abdominal reflexes have not been consistently absent in this patient, although they usually disappear in multiple sclerosis. Furthermore, it is unlikely that demyelination of the white matter would persist and progress for 20 years without showing typical lesions of disseminated sclerosis.

The appearance of convulsions 18 years after the acute onset of encephalitis is an unusual feature of this case. The electro-encephalography showed abnormal waves in the frontal area of the brain. Such irritative foci indicate that brain damage has occurred here. From a

clinical standpoint the convulsions were Jacksonian in character and seemed to originate in the right motor area. As yet an encephalogram has not been made, but may be indicated if the attacks are not controlled by dilantin and phenobarbital.

CONCLUSION

The progress of this patient during the past 20 years has manifested signs and symptoms of a rather diffuse and progressive lesion of the central nervous system, yet he has not shown any clinical manifestations of degenerative changes in the basal ganglia. The neurological changes here have been difficult to place in any definite classification. If a history of acute encephalitis had not been obtained from this patient, we would have regarded him as having atypical multiple sclerosis.

It is of further interest to record that he has worked quite steadily during this long period with little loss of time, and it appears that he will lose only five or six weeks of working time in this present episode. With reference to treatment we doubt that the administration of arsenic and iodides had any definite influence upon the course of the disease.

However, in the past month he has improved markedly with the use of dilantin not only in the control of his convulsions but also in a general feeling of well-being.

Regardless of what the future has in store for this patient, it is fortunate that his defense mechanisms were adequate to keep this disease in abeyance for so many years.

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POOR POSTURE AND LOW BACK PAIN

W. J. ZEITER, M.D. and G. J. WARD, M.D.

Clinicians frequently see patients who are obviously in poor health, but in whom no organic disease can be demonstrated. In a large number of these chronically ill patients, poor body mechanics may be playing an important role in the chronic disability, and beneficial effects may be obtained when the error in mechanics is improved or entirely corrected.

The White House Conference on Child Health[1] defines body mechanics as follows: "Body mechanics is the mechanical correlation of the various systems of the body with special reference to the skeletal, muscular, and visceral systems and their neurological associations."

In the following discussion the effects of poor body mechanics shall not be described in detail, but rather the emphasis shall be upon the essential changes that are produced and upon the methods for correcting faulty posture.

Every clinician has noted that the individual who stands with erect posture feels alert, looks alert, and portrays energy and a sense of well-being; whereas, the slumped individual with a marked forward curve of the cervical spine or increased lordosis makes the opposite impression. In our armed services where much attention is given to the posture of the fighting man, the value of good posture is recognized. If the civilian population were more posture conscious, they too would be benefited.

Because of the differences in body builds, each patient's problem must be individualized. Accordingly, no scale can be set up which will be adaptable to every patient. As Goldthwait[2] has so aptly stated, when a mechanical engineer studies a machine he first looks at it to see how it is made, and then examines it to see how near to 100 per cent it is in alignment and function. The less well the machine is aligned, the greater is the strain and the potential of trouble. Likewise in the human machine malalignment or faulty mechanics are potentials of trouble and should be recognized by the physician as such. In order that the various complicated systems may function with the least amount of wear and the greatest amount of power, the body should be developed to its highest mechanical efficiency.

The bones, ligaments, and muscles determine the range of motion of a particular joint. Faulty body mechanics may produce a long series of anatomical malalignments which if allowed to progress can result in severe visceral, somatic, and neurological symptoms. In the bony

structure of the vertebral column there are certain curves, and all the vertebrae have a certain range of motion. If these curves are accentuated, strain is placed upon the articulating facets, ligaments, and musculature. With marked accentuation pressure may be produced on the peripheral nerves as they leave the spinal column. The site of pain depends upon whether the maximum malalignment is in the upper or lower spine. Occipital headaches or pain in the neck not infrequently result from faulty mechanics of the cervical spine.

The greatest flexibility of the spine is in the region of the dorsolumbar segments, and because of this greater flexibility mechanical error often occurs at this site. Probably the most common clinical problem from chronic postural strain is low back pain. The exercises which shall be described later in this discussion are directed primarily toward the correction of mechanical strain producing low back pain in which organic disease has been ruled out.

Pressure on the nerves in the dorsolumbar region may produce referred pain in the region of the gallbladder, appendix, or lower abdomen. After organic disease has been ruled out, attention should be directed to postural defects as a possible etiological factor.

Because of the attachment of the ribs to the dorsal vertebrae, an increased dorsal curve will produce a downward inclination of the ribs, thereby flattening the chest to more closely approximate the ribs and to greatly decrease the depth of the chest, which in good body mechanics is about two-thirds of its width. This condition is more likely to occur in the slender type of individual. With the flattening of the chest the abdominal muscles become relaxed and less efficient in supporting the abdominal viscera. As the circumference of the bony thorax is diminished, the origins and insertions of the diaphragm are more closely approximated with a resultant diminution in efficiency. The reserve function for unusual exertion such as is needed in singing is also decreased since the diaphragmatic excursion in the standing position is at, or very near, the point of full inspiration. Because the great abdominal veins are attached to, and pass through the diaphragm, the diaphragm has an additional function of aiding the return of the blood to the right heart. Accordingly, the importance of good posture and breathing exercises should be emphasized in disease in which maximum circulatory and respiratory efficiency is desired.

When the position of the diaphragm is low and the abdominal muscles relaxed, the abdominal cavity alters its shape from the ideal. In the ideal position the abdominal cavity is largest in the upper portion and decreases in size in the lower portion, assuming a pear shape with the top of the pear pointing downward. If this position is assumed,

the abdominal muscles evidence good tonus and help to keep the abdominal viscera elevated. If the abdominal muscles are relaxed, the opposite occurs.

Before measures for the correction of faulty body mechanics can be applied, the various gradations in body carriage must be understood. The Children's Bureau of the U. S. Department of Labor[3] directs attention to these variations and describes three types of anatomic structure.

1. "The thin type has a torso long and slender or delicate and narrow and a long, thin neck. The length in the lumbar region is striking. Frequently there may even be six lumbar vertebrae instead of the usual five. The elongated spine gives more flexibility, and this explains the marked slump, or ptosis, possible in these persons. They sometimes look as if some heavy force were pushing them down from above; so much so that they sway far backward in the upper back and protrude far forward in the lower back, the pelvis tipping forward in the lower spine. The shoulders may become markedly rounded and forward and the shoulder blades scaphoid. The extremities and their muscles are usually long and slender.

2. "The broad type includes the heavy-looking, 'broad-backed' persons with large skeletons. The neck is short and 'chunky.' The torso is broad and relatively short. The lumbar region is short, sometimes because there are only four lumbar vertebrae instead of the usual five and sometimes because the sacrum is set well down between the hip bones. Because of the very construction of the spine the lumbar curve is less marked. Flexibility is lacking in this sturdily built spine. The extremities are large and broad. This is the type that tends to be obese.

3. "In the intermediate type the torso is a compromise in length and breadth between the other two types. The normal rounded curves of the spine, if they become exaggerated, appear mild and gradual. The sharp 'corners' of this thin type and the large fatty deposits of the broad type are missing. The neck may be almost as long as that of the thin type; or it may be short, though hardly so thick and 'chunky' as in the broad type. The musculature is firm. Flexibility of the spine, though not so marked as in the thin type, is much greater than in the broad type. This intermediate class is heterogeneous; it should include all individuals that do not fall readily into either of the other groups."

Various methods have been suggested for the correction of faulty body mechanics, and the following exercises describe the method we use to correct mechanical strain, especially involving the low back.

The exercises are taught in two positions: (1) in the recumbent position, and (2) in the erect position. In the preceding paragraphs

emphasis has been placed upon the importance of chest expansion and satisfactory range of motion of the diaphragm. It is well therefore to begin with the breathing exercises which can best be done on a firm treatment table which is perfectly flat.

Exercise 1. The patient lies flat on the back without a pillow under the head. The knees are flexed with the feet flat on the table. The fingers are inserted into the costal margin to assist in raising the costal margins in deep inspiration. Inhalations are taken through the nose and exhalations through the mouth, and an attempt is made to expand the thoracic cage to its maximum limits.

Exercise 2. After the above exercise, diaphragmatic breathing is developed. This is accomplished by attaining complete expansion of the chest. Respiration is continued by diaphragmatic movement while the chest is held expanded.

Exercise 3. Pelvis tilting. The knees are flexed with the feet flat upon the table to help eliminate the lumbar curve so that it approximates the table. The knees are kept in the flexed position, and the pelvis is rolled forward to produce a lordosis and then rolled backward to eliminate the lumbar curve. The motion primarily involves the gluteal and abdominal muscles which are of prime importance in maintaining the proper pelvic angle.

Fig. 1

Exercise 4. Head raising. (Fig. 1) With severe lordosis head raising is sometimes necessary to assist in the correction. The knees are kept in the flexed position, and the arms are extended so that they cannot be used for support. Then the head is raised from the table.

Exercise 5. Pelvis tilting with gradual extension of the legs. The pelvis is tilted with the knees flexed and the feet flat on the table to eliminate the lumbar curve as in Exercise 3. The heel of the right foot is kept in contact with the table, and the leg is gradually extended until flat on the table. The same procedure is then carried out with the left leg. During this exercise every attempt is made to keep the lumbar spine flat.

Exercise 6. The supine position is assumed with pelvis tilted to flatten back as in Exercise 3. The right leg is gradually extended until it is flat on the table. The leg is then flexed upon the trunk to the maximum degree while the knee is kept straight and all motion of the limb occurs at the hip. The leg is then brought back to the table. The same procedure is repeated with the left leg.

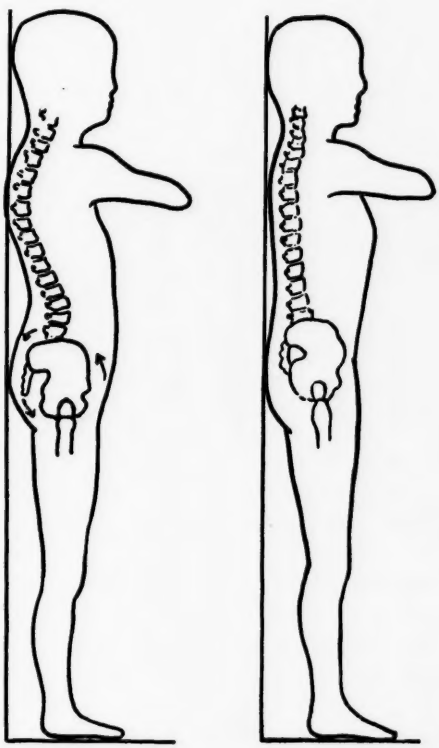

Fig. 2

Exercise 7. (Fig. 2) Pelvis tilting and flattening the back against the wall. The patient stands with the heels 4-6 inches from the wall. The buttocks, shoulders, and head touch the wall, and the chin is held at a right angle. The patient then elevates the symphysis upward, which rotates the pelvis backward thus reducing the lumbar curve, and tries to press the lumbar spines against the wall.

Exercise 8. After the position has been assumed properly as directed in Exercise 7, the patient is instructed to walk forward two or three paces, rise on the toes several times, and return to the wall. During this maneuver the patient is attempting to maintain the position achieved by Exercise 7. The return to the wall acts as a check upon the degree of correct posture maintained during the simple process of walking.

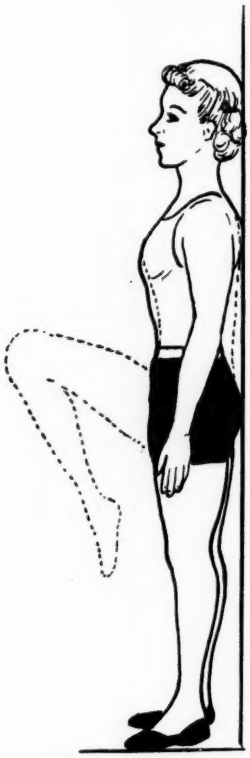

Fig. 3

Exercise 9. The patient assumes the position as in Exercise 7. The right knee is flexed upon the abdomen, as in Fig. 3, and this position is exaggerated by grasping the knee with both hands and pulling it toward the abdomen while the lumbar spine is simultaneously pressed against the wall. The leg is gradually lowered to the standing position, and the position of the lumbar spine is maintained as achieved with the leg flexed. The same procedure is carried out with the left leg.

Exercise 10. This exercise consists merely in walking about for a few minutes and maintaining a flattened lumbar curve. This maneuver is primarily to acclimate the patient to the new and as yet unfamiliar position.

Exercise 11. The patient stands against the wall and again assumes the correct posture as in Exercise 7. He then flexes the spine and allows the arms to drop forward loosely, and then straightens up having each vertebra touch the wall beginning in the sacrum and progressing up the spine.

The patient executes each exercise about ten times unless there is marked weakness or fatigue which would limit the number of times the procedure is repeated.

The described exercises are directed toward correcting mechanical error especially of the dorsolumbar region. In some instances more elaborate and detailed exercises are needed to correct malposition. The correction of faulty body mechanics requires more than a few minutes a day spent in performing exercises. These exercises will give tonus to those muscles instrumental in maintaining good posture. However, the patient must become posture conscious and make a wilful effort throughout the day to assume the posture reached in his exercises, and attempt to fulfil the ten commandments of good posture as suggested by Lewin.[4]

1. Stand tall.
2. Sit tall.
3. Walk tall and "chesty" with weight transmitted to balls of feet.
4. Draw in abdomen, pulling it backward and upward.
5. Keep shoulders high and square.
6. Pull chin down toward collar button.
7. Flatten hollow of back by rolling pelvis downward and backward.
8. Separate shoulders from hips as far as possible.
9. Lie tall and flat.
10. Think tall.

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THE MANAGEMENT OF GRASS HAY FEVER

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In the greater part of the United States three distinct hay fever seasons are recognized, which correspond to the pollination periods of three plant groups, trees, grasses, and weeds. In the northern states the grass (early summer) hay fever season extends from the last of May until the latter part or the end of July; in the beginning it parallels the late stages of the tree hay fever season.

The grasses are the most widely distributed of the three groups of plants producing hay fever. As a rule they produce a less severe form of hay fever than the weeds, but are decidedly more important than the trees. Unlike the weeds, however, the grasses are important commercially and include the pasture grasses and cereal grains. In the northern states timothy and June grass, two important pasture grasses, are of primary importance in the production of grass hay fever, whereas, in the southern states Bermuda grass is the chief agent. In the northern states the grass hay fever season corresponds to the combined pollination periods of June grass and timothy. Orchard grass and red top are of secondary importance. The cereal grasses are of minor significance except in extensively cultivated areas where rye grass may be of some importance.

Although the term "rose fever" is commonly used by the laity to describe grass hay fever, garden flowers seldom produce symptoms. Flowers are insect pollinated, the pollen is heavy and sticky, and is not sufficiently abundant to be a significant cause of hay fever. Close contact with flowers may produce hay fever symptoms in certain individuals, but even in these cases grass pollen adhering to the rose or other flowers may be the actual cause of the symptoms. However, it is wise to advise hay fever victims to avoid close contact with flowers during the hay fever season.

Although the diagnosis of hay fever is comparatively easy to make, a careful history is advisable and may reveal much significant information. The seasonal recurrence of attacks of sneezing, nasal obstruction, rhinorrhea, and lacrimation are pathognomic. Nasal symptoms usually predominate, but at times the ocular symptoms are very annoying. A dry irritative cough may develop which may indicate incipient asthma. Asthma is the most important complication of hay fever and ultimately develops in about one-third of the untreated cases.

The initial attack of hay fever may be confused with a common cold. After the first day or two of symptoms the thick purulent secretion of the common cold is in marked contrast to the thin watery secretion in the

hay fever patient. The pale edematous turbinates in the allergic individual are also in marked contrast to the reddish, inflamed, mucous membranes in the patient with a cold. A predominance of eosinophiles in the nasal smear also indicates the allergic cases.

Although the great majority of patients consulting us with hay fever are otherwise healthy, a complete physical examination is made in all cases. During the hay fever season while the patient is having symptoms, typical conjunctival injection with lacrimation, rhinorrhea, and pallor and edema of the nasal mucous membrane are noted. A careful chest examination is always made in an effort to detect incipient asthma. Routine laboratory procedures including urinalysis and complete blood counts are also thought to be a worthwhile part of the examination.

A complete allergy investigation should be carried out not only to confirm the diagnosis but also to detect other allergic factors which may be aggravating the patient's symptoms. The date of onset and offset of symptoms should indicate the offending pollen group (grasses or weeds), but the detection of other inhalant or food allergens may result in a much more satisfactory response to treatment. We test routinely for the more common tree, grass, and weed allergens and make about 42 pollen tests in all. These pollen tests are made by the scratch method. Scratch tests are also used for the usual inhalant and food allergens, and all negative scratch tests are rechecked by the endermal method. In the occasional case in which pollen tests are negative or doubtful, and the patient apparently has hay fever, ophthalmic or nasal contact tests are valuable.

Usually patients with grass hay fever react not only to one but to a number of grasses. Fortunately a common antigenic relationship is believed to exist among the various members of the grass family, and for this reason timothy extract is frequently employed as hyposensitization for all the grasses. Better results, however, are obtained by using a mixture of the grasses. Our practice is to use an extract of the four most common grasses in this locality. We have found a mixture of timothy 50 per cent and red top, June grass, and orchard grass 50 per cent to be quite satisfactory. The accepted method for treating hay fever is specific pollen hyposensitization which attempts to increase the patient's tolerance to pollen by injections of gradually increasing amounts of pollen extract. At the Clinic we use all three methods of hyposensitization, preseasonal, coseasonal, and perennial. For the best results in the average case, we prefer the perennial method.

The preseasonal method of hyposensitization was the first to be used and is the one most frequently employed. Ideal treatment is begun about three months prior to the expected onset of symptoms. The first dose is small, the dosage increments are small, and the maximum dose should be reached at the time of onset of pollination. The initial dose is usually

1/10 cc. of a 1:5,000 concentration of the pollen extract. Injections are given twice weekly and are increased by 1/10 cc. provided, of course, that it is well tolerated. After reaching a dosage of 1 cc. of a 1:5,000 concentration, injections are continued with a 1:500 dilution beginning with 1/10 cc. and increasing as before. After the 1 cc. dose of 1:500 dilution is reached, the 1/50 dilution is used similarly.

In the average case we advise about 0.4 cc. of the 1:50 dilution (8000 Noon units) as the maximum dose which should be reached by the last week in May. A maintenance dose of approximately ¼ to ½ the maximum dose (usually 0.1 cc.) is then administered every two weeks until the latter part of July, at which time grass hyposensitization is discontinued. Treatment is resumed on March 1st, the following year.

The dosage schedule may be modified to suit the individual patient's needs. In some instances the maximum dose which the patient will tolerate is 2000 Noon units or less. Very satisfactory relief may be afforded by such a dose. When the time available for treatment is short, the schedule may be speeded up somewhat, omitting some of the doses. When the onset of the hay fever season is less than seven weeks distant, however, it is better to withhold preseasonal treatment.

Coseasonal treatment attempts to relieve symptoms by the administration of small doses of pollen extract as frequently as necessary and should be started as soon as the first definite symptoms are noted. It may be used in patients who have had no therapy prior to the onset of the season or as a supplementary measure whenever preseasonal or perennial treatment has not given satisfactory relief. It is especially helpful when preseasonal treatment has been inadequate either because of a late start or irregular treatment.

Coseasonal treatment is begun as soon as symptoms become troublesome. The first dose given is very small, usually 0.1 cc. of the 1:5,000 pollen extract. If satisfactory relief is obtained, the same dosage is continued upon return of symptoms. If relief is inadequate, the dosage of the second injection is increased. Whereas, if the first injection causes an exacerbation of symptoms, the second dose is reduced. The injections are continued daily or every second or third day depending upon symptoms, and the dosage may be gradually increased consistent with the symptomatic relief obtained. The maximum dose reached during the season seldom exceeds 0.5 cc. of the 1:5,000 pollen extract. It is wise to supplement the pollen treatment with 2/10 cc. of 1:1,000 adrenalin solution or preferably a mixture of adrenalin 1:1,000 and ephedrine 3 per cent in equal parts.

Perennial therapy is looked upon with favor by most allergists and is an attempt to maintain the patient's tolerance to pollen at a high level

throughout the year with hope of producing a permanent remission. A so-called permanent "cure" does occur in a small percentage of cases. It is debatable whether perennial therapy gives more adequate seasonal relief than the preseasonal method, but it has the advantage that the patient is kept under supervision throughout the year which permits the earlier detection of other allergic manifestations such as the development of a perennial rhinitis. Furthermore, it avoids intensive preseasonal treatment, and spring vacation or illness will interfere much less with perennial than with preseasonal treatment.

Perennial therapy may be instituted any time during the year, preferably as soon as the diagnosis is made. If sufficient time is available before the expected onset of the hay fever season, the injections are given only once a week in dosage similar to preseasonal treatment. Upon reaching a dose of 1/10 cc. of the 1:50 grass mixture, injections are maintained at that level and the interval is extended to semi-monthly. About May 1, or approximately four weeks before the expected onset of the grass season, the weekly schedule is resumed, and the dosage is increased 1/10 cc. weekly reaching the maximum dosage of 0.4 cc. of 1:50 dilution by the end of May. Thereupon a maintenance dose of 1/10 cc. is resumed twice monthly and continued throughout the remainder of the year. The hyposensitization injections are similarly built up prior to the onset of the season the following year.

Although the treatment of choice is specific pollen hyposensitization, a few fortunate individuals avoid pollen contact each year by moving to pollen free districts. Air conditioning or a mechanical filtration of the air, either of the whole or part of the home, will provide adequate relief, but this restricts the patient's activities to such an extent that it is applicable to only a few. Such palliative or symptomatic measures are helpful in cases otherwise untreated, or in which inadequate relief has been obtained. Symptoms will be lessened by keeping off of golf courses and avoiding open windows and traveling during the season, especially by automobile. Any form of strenuous exercise such as swimming and tennis is also to be avoided. Dark glasses partially relieve ocular symptoms, and eye washes or preferably cold compresses of boric acid or normal saline are recommended. When ocular distress is marked, a weak solution of adrenalin chloride (1:5,000 or less) in normal saline is of considerable value. Ephedrine in dosages of gr. ⅜ preferably combined with amytal gr. ¾ or phenobarbital gr. ¼ will relieve some of the nasal distress. If rhinorrhea is very marked, atropine sulfate in dosage of 1/300 to 1/500 gr. may be used in addition to the above. Nasal sprays of ephedrine 1 – 3 per cent or neosynephrine ¼ to 1 per cent are also recommended.

CASE REPORTS

The following two case histories illustrate a typical problem of grass hay fever and another type of seasonal rhinitis which may be confused with hay fever.

Case 1. A 15 year old youth had had the typical ocular and nasal symptoms of grass hay fever for two years. His symptoms began approximately May 30 and were worse during June and July. His symptoms did not entirely clear up until frost, although there was no flare-up during August or September to suggest weed hay fever. He had no asthma.

The history further revealed one severe attack of hives and one attack of poison ivy. His father had had both grass and ragweed hay fever and had developed perennial asthma seven years prior to death which had occurred during a severe asthmatic paroxysm. One brother had grass hay fever and urticaria.

The physical examination was normal except for pallor and edema of the nasal mucous membranes with slight conjunctival injection. Routine laboratory examinations were normal except for a 6 per cent blood eosinophilia. Sensitization studies revealed definitely positive reactions to all grasses as well as to the pollen of the grain, rye. There were no weed reactions. Several reactions to inhalants were obtained, the more important of which were to house dust, orris root, and molds. Food reactions included wheat, corn, rice, chicken, peas, beans, and sweet potato.

The patient was first seen in September. Preseasonal grass treatment was started the next spring approximately March 1 according to the plan outlined and reached the maximum dose in the latter part of May. At the same time an inhalant extract of the more important inhalant allergens was given, and he was advised to avoid unnecessary contact with the various inhalant allergens and to follow a restricted diet during the hay fever season. He obtained excellent relief and was almost 100 per cent free of symptoms during the entire hay fever season.

A second case illustrates seasonal rhinitis from mold sensitivity simulating hay fever. This type of problem might be called non-pollen hay fever.

Case 2. A 14 year old girl suffered for five consecutive summers from attacks of sneezing, nasal obstruction, and watery discharge. The symptoms developed in early June and disappeared following the first frost. The year preceding treatment, however, they had started in late May, for the first time. She was free of symptoms during the winter months and had only the usual number of colds. No other allergic manifestations had been noted. Her father had a chronic catarrh, and her mother had migraine.

The physical examination and routine laboratory investigation were essentially normal.

The few pollen reactions obtained were questionable. Ophthalmic tests for both grass and weed pollens revealed only questionable reactions to the dry powder. Endermal tests to inhalants, however, revealed positive reactions to house dust, feathers, orris root, and to several molds, notably a strong reaction to alternaria. Several positive food reactions were obtained, chiefly to the bean group.

An avoidance program for the inhalant and food allergens was outlined, and hyposensitization for the significant inhalants, including molds, was instituted. Treatment was begun on April 15. Mild nasal symptoms developed on May 23. By July 1 these symptoms were much less severe than during the previous year and remained fairly well controlled all summer. When last heard from on June 13 of the following season, she had had several weeks of treatment and was entirely symptom free.

The first of these cases represents a typical case of grass hay fever, and the second, a type of seasonal rhinitis which may be confused with

pollen hay fever. It is worth observing that from the history alone a case of seasonal rhinitis due to molds was not distinguishable from symptoms produced by pollinosis. This emphasizes the value of complete allergy investigation in such cases. Seasonal rhinitis due to factors other than pollens usually is associated with symptoms not sharply limited to any one of the hay fever seasons. Symptoms usually begin late for the grass hay fever season and early for ragweed. Furthermore, exacerbations of symptoms do not correspond to peaks in the pollen count.

In cases of mold allergy symptoms are usually worse following damp periods and improve in dry weather, whereas, in hay fever the opposite holds true. Control of symptoms by hyposensitization with mold extracts confirms the diagnosis.

SUMMARY

No attempt has been made to discuss all aspects of the grass hay fever problem. The emphasis has been upon treatment, and various types of hyposensitization have been discussed. Our method of treatment has been given in some detail, and our reasons for preferring perennial therapy.

Certain general conclusions may be emphasized.

1. Each case of hay fever must be treated as an individual problem and warrants complete investigation.

2. A detailed history is extremely valuable and should include an investigation of other manifestations of allergy, especially as to whether or not asthma exists as a complicating factor.

3. Pollen tests alone are not adequate and should be supplemented by tests with the more common inhalant and food allergens.

4. Allergy management should include not only specific pollen hyposensitization, but also the avoidance of other significant inhalant and food allergens and hyposensitization for other inhalant allergens, especially molds, when indicated.

We believe that the treatment of choice is perennial pollen therapy, supplemented by a well-rounded program of allergy management, which may also include coseasonal pollen treatment if symptoms develop to warrant it.

THROMBO-ANGIITIS OBLITERANS

FAY A. LeFEVRE, M. D.

Thrombo-angiitis obliterans is an organic disease which involves the arteries and veins. It is inflammatory in nature and usually involves the peripheral vessels. Vasospasm secondary to the organic process is a characteristic feature of the disease.

Von Winnwater originally described this condition in 1879 and termed it endarteritis obliterans. In 1908 Leo Buerger[1] gave a more accurate description of the disease and called it thrombo-angiitis obliterans. Dr. Buerger's work initiated further study by numerous investigators, but up to the present time the etiology has not been determined. Many believe that Buerger's disease is a specific inflammatory process which is the result of some bacterial or virus invasion. Conclusive proof of this contention has not been demonstrated. The difficulties involved in making experimental studies on patients with this disease are obvious.

Thrombo-angiitis obliterans is one of the most destructive of all peripheral vascular diseases. Because it occurs in young individuals and may result in a physical handicap, an early diagnosis is of extreme importance. If the diagnosis is made early and proper treatment instituted, the course of the disease may be checked. If the patient is seen too late or if the diagnosis is delayed, surgical removal of the limb may be the only form of treatment available.

Diagnosis: Thrombo-angiitis obliterans should not be difficult to diagnose if several distinguishing and characteristic features are remembered. The disease occurs almost exclusively in young men, and the ratio of men and women is thought to be at least 75 to 1. The age incidence varies, but the great majority of cases occurs between the ages of 25 and 45. In a patient over 50 years of age the diagnosis should be made with great caution. Although 50 per cent of patients observed are Jews, any race may be subject to the condition.

The vessels chiefly and initially involved are those of the lower extremity, and the symptoms and signs at first are unilateral. The disease usually progresses from one extremity to the other, and in a large percentage of cases the arms may become involved. Typical lesions of the cerebral, coronary, and mesenteric arteries have been reported.

The clinical course frequently is characteristic. Accurate histories indicate that the disease is definitely intermittent in nature. A patient seen for the first time should always be questioned for a history of

phlebitis. Although this episode of phlebitis may have occurred years before the present symptoms, it may have been the actual onset of the disease.

Periods of arterial or venous involvement are usually followed by a remission of symptoms. During this time collateral circulation may form, and the symptoms of occlusion may be relieved. As the disease progresses, the degree of closure usually becomes more pronounced. Usually the patient is seen within two years after the onset of the disease, which may run an intermittent course of five to 15 years. Occasionally in acute cases some changes develop within a few months.

If it is remembered that thrombo-angiitis obliterans is a progressive intermittent disease producing various grades of ischemia, the resulting symptoms and signs can be easily explained. The earliest symptom is usually a sense of cold in one or more extremities which may be present for several years. Frequently the patient observes that the feet have changed color and have a ruborous appearance especially in dependent positions. Mild cyanosis is also present at times. The presenting symptom, however, is pain, which at first may be described as fatigue and in early stages may be mild and observed only after strenuous effort. This pain, or fatigue, usually occurs after the patient has walked a certain distance and is relieved by rest. This intermittent claudication generally involves the calf area, but may involve the foot and entire leg. Examination at this time reveals color and temperature changes. One foot may be definitely colder to the touch than the other. Also, the peripheral pulses may be absent or diminished on palpation. These changes may be slight, and special temperature and oscillometric studies may be necessary to determine the presence of occlusion.

As the degree of occlusion progresses, the ischemia becomes more marked; the pain becomes constant and often is worse at night. The color and temperature changes are more marked, and trophic changes such as ulceration may involve the terminal portion of one or more toes. Arterial thromboses occur and cause very severe pain. The patient is unable to sleep, becomes nervous, and smokes heavily, which in turn increases the vasospasm and produces more severe ischemia. If the disease progresses, true gangrene results with necrosis and spread of infection. Severe toxemia may make amputation necessary.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of thrombo-angiitis obliterans should not be difficult if the aforementioned clinical features are kept in mind. The two common conditions with which it might be confused are arteriosclerosis obliterans and Raynaud's disease. The chief points of differentiation are summarized in Table I.

Treatment: If the presence of thrombo-angiitis obliterans is suspected, the patient usually should be hospitalized in order to permit a complete study of the case. Proper treatment also can be started without delay, and the patient can be informed of the true nature of his disease. Horton[2] has stated: "It is just as important to educate the patient who has thrombo-angiitis obliterans regarding the nature of his disease as to instruct a patient who has diabetes regarding his diet." In addition, complete bed rest is a very important part of treatment, particularly if the disease has progressed beyond the stage of simple intermittent claudication, and is fundamental if there are any trophic changes.

Whether or not the patient goes into the hospital, a careful and thorough physical examination with complete blood studies should be made. The presence of syphilis, diabetes mellitus, and polycythemia vera must be excluded. Also, special tests such as temperature readings and oscillometric readings may be made, and the degree of organic and spastic involvement determined.

After the diagnosis has been established, the patient first should be instructed in the general care of the extremities. (Table II)

TABLE I

Differential Diagnosis

	Thrombo-Angiitis Obliterans	*Arteriosclerosis Obliterans*	*Raynaud's Disease*
Sex	Men—99%	Men—90%	Women—95%
Age	25–45	50 and over	15–35
Race	Any–50% Hebrew	Any	Any
Tobacco	Large amounts	Moderate	Not frequent
Type of pain	Severe and sharp	Dull—aching	Frequently absent
Location	Unilateral—any extremity	Bilateral—usually lower extremity	Bilateral—upper extremity
Color changes	Rubor and pallor	Same	Cyanosis and pallor
Type of ulcer	Inflamed—moist	Dry	None
Gangrene	Common	Common	Rare
Pulses	Absent	Absent	Normal
Phlebitis	Common	Absent	Absent

TABLE II

General Directions for Home Care of the Feet

The following instructions are outlined for patients with circulatory disturbances of the extremities. The instructions should be followed carefully, as they constitute an important part of the treatment.

1. Do not use tobacco in any form.
2. Drink at least four quarts of water daily unless specified not to by doctor.
3. Eat a normal, well-balanced diet unless ordered to follow a special diet.
4. The following routine should be carried out every night:

   Wash feet every night, using warm (not hot) water and neutral (face) soap. Dry feet with a soft towel. Dry carefully between toes. Do not rub feet.

   Apply 70 per cent rubbing alcohol and a liberal amount of lanolin or 5 per cent boric acid ointment. Never apply strong antiseptics such as iodine or lysol to feet. If ulceration is present changes in this routine will be made by the physician.

   Cut toe nails after feet have been cleansed and always cut straight across and not too short.

   Do not cut corns or callouses at any time.

5. Keep feet warm at all times. Wear proper shoes as directed. Wear protective stockings and warm under-clothing. Wear loose-fitting bed socks at night. Never apply a hot water bottle or electric pad to feet or legs. Avoid exposure to cold weather.
6. Do not wear circular garters or sit with legs crossed.
7. Carry out the leg exercises as directed.
8. Special instructions:—

DO NOT ATTEMPT TO TREAT BLISTERS, INGROWING TOE NAILS OR INFECTIONS OF THE TOES AT HOME. IF ANY OF THESE CONDITIONS DEVELOP, NOTIFY YOUR PHYSICIAN AS THIS MAY BE THE FIRST SIGN OF SERIOUS ULCERATION AND GANGRENE.

Although the treatment cannot be standardized for every patient, the principles outlined apply to the average case, and changes may be made whenever necessary. All patients are advised to discontinue the use of tobacco in any form. Although smoking may not be the cause of this disease, it certainly plays an important part in the patient's recovery. Frequently, patients show spontaneous improvement when smoking is discontinued. In my own experience, a case of thrombo-angiitis obliterans has never improved until the patient has discontinued smoking.

As has been previously mentioned a period of bed rest for all patients with extensive involvement should be prescribed. During this time heat should be applied with extreme care. I use a thermostatically-controlled cradle which is placed over the lower extremities and maintained at a temperature of about 96 degrees F. This eliminates the possibility of burns such as may occur from the use of a heating pad or a hot water bottle. It is my impression that diathermy should not be used in any case of thrombo-angiitis obliterans.

Buerger-Allen exercises are prescribed routinely and should be done at least twice daily for an hour at a time. In the average case they consist of elevating the feet at an angle of 60 degrees for three minutes, allowing the feet to hang over the side of the bed for three minutes,

during which foot exercises are done, and finally resting for three minutes at bed level. This procedure may be repeated six times during the hour. The periods of elevation and dependency frequently have to be changed in accordance with the degree of vascular occlusion. Otherwise, pain may be experienced, especially in the elevated position.

In my experience, the intravenous administration of typhoid vaccine is the most satisfactory form of medical management. I prefer to use relatively small doses of typhoid vaccine and make a definite attempt to avoid high elevations of temperature. The usual initial dose of 5 million bacteria is given intravenously, and the temperature reaction is observed. An attempt is made to elevate the temperature about 2 degrees F. After the temperature has been normal for about 24 hours, a second injection is given. The dosage may be increased in order to maintain a 2 degree rise in temperature and on an average ranges between 5 million and 20 million bacteria. During a two-week period of hospitalization approximately eight injections may be given, and after a period of rest of from three to four weeks the course may be repeated. Following the injections a definite vasodilatation occurs for a period of from six to eight hours. If the fever rises too high, thrombophlebitis may occur. Typhoid therapy does not benefit all patients suffering from thrombo-angiitis obliterans. Patients having rest pain and early trophic changes usually show the best response. In the presence of definite, regular, intermittent claudication less benefit may be expected. When ulceration and gangrene are advanced, typhoid therapy is of very little value, but may be used as a general measure to help arrest the progress of the condition.

Numerous other medical measures have been advised for the treatment of thrombo-angiitis obliterans. The use of hypertonic sodium chloride solution administered intravenously has been advised by many. I continue to use this type of medication, but do not think its results are as satisfactory as those from the typhoid routine. In some cases it is used to supplement the typhoid vaccine regimen. The administration of tissue extracts and acetyl-β-methylcholine (mecholyl) by iontophoresis also has been recommended. My experience with these methods has been generally unsatisfactory. The sulfonamide drugs seem to produce some benefit, particularly if there is evidence of extensive infection. I recommend their use routinely, particularly in the presence of ulceration and gangrene when the process is extending rapidly.

During the past few years the use of heparin has been tried. This is, of course, given intravenously with the thought of preventing extensive thrombosis. More recently an oral preparation known as [3,3′-methylene-bis-(4-hydroxy coumarin)] has been tried. This product

has an effect similar to that of heparin on the prothrombin time and coagulation time. It is too early to evaluate the results of this type of therapy.

Except for the controlled use of heat and the Buerger-Allen exercises, physical therapy measures have not been found to be of particular benefit. The positive and negative pressure apparatus, which is of value in cases of arteriosclerosis obliterans, does not seem to be helpful in cases of thrombo-angiitis obliterans. My experience with the intermittent venous occlusion apparatus has been limited and to date does not indicate that it is any more beneficial than the positive and negative pressure apparatus.

Sympathetic ganglionectomy may be valuable in properly selected cases. The operation does not cure the disease, nor does it alter the course of the disease in the blood vessels, but to a certain extent it does relieve any secondary vasospasms and may allow a maximum blood flow. Before the operation is performed, temperature studies should be made to determine definitely the degree of vasospasm. In the presence of a vasospastic element the procedure should be considered seriously.

It is always difficult to determine whether or not a surgical amputation should be performed. The natural tendency is to delay and to try every possible conservative measure before resorting to amputation. If the area of ulceration is not extensive and if there is a definite line of demarcation, it is my belief that amputation should be postponed and an attempt made to restore the circulation. If the disease is progressing rapidly and there are definite signs of toxemia, surgical amputation at a safe level should not be delayed.

SUMMARY

Thrombo-angiitis obliterans is an organic disease of the arteries and veins which causes extensive damage unless its progress is checked in the early stages. The disease usually affects men between the ages of 25 and 45. It is not confined to any racial group, although Jews are more susceptible. An early diagnosis is exceedingly important and is one of the factors most essential for successful treatment. The patient should be thoroughly familiar with the nature of his disease so that he may cooperate in protecting himself from the dangers which may ensue. The use of typhoid vaccine intravenously is suggested as a satisfactory form of medical treatment. However, general measures are equally important and probably benefit the patient more than any specific form of treatment.

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